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Identification of Childhood Arthritis in Archaeological Material: Juvenile Rheumatoid Arthritis Versus Juvenile Spondyloarthropathy

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KEY WORDS growth retardation; bone erosion; pseudoarthrosis; pathology

ABSTRACT The opportunity to examine the defleshed skeleton of an individual diagnosed in life (Hamann-Todd collection, individual 2036) afforded a unique opportunity to demonstrate the bone damage characteristic of at least one form of juvenile rheumatoid arthritis (JRA). Characteristics helpful for recognition of JRA in archaeological material include peripheral articular marginal and subchondral erosions, axial (e.g., zygapophyseal or sacroiliac) joint erosions, fusion of axial (cervical zygapophyseal) and/or peripheral joints, premature epiphyseal closure and/or ballooned epiphyses, growth retardation with underdeveloped (short and overtubulated) long bones, short mandibular rami with underdeveloped condyles and concomitant micrognathia, and demineralization (osteopenia). Distinguishing between JRA and juvenile spondyloarthropathy, however, is not always possible, as illustrated by this case. Am J Phys Anthropol 102:249–264, 1997

A major obstacle to recognition of childhood disease in archaeological material is paucity of reliable diagnostic criteria. Given the rare availability of defleshed skeletons (diagnosed in life) of children, even isolated cases are important to allow development of criteria for disease recognition. While not affording the power of quantitative population study (Ortner and Putschar, 1981; Rothschild and Martin, 1993), study of individual skeletons allows qualitative information.

Although there are numerous clinical publications on juvenile rheumatoid arthritis, there are no macroscopic descriptions of the bony lesions associated with it and only one report of a presumptive case in the archeologic record (Buikstra et al., 1990). Relying on radiological studies is problematic, because many of the osseous changes are nonspecific, and many anatomical details are beyond x-ray resolution. The study of a defleshed skeleton with documented JRA should facilitate identification of such childhood diseases in the archaeologic record.

Although arthritis is not common among children, appearing in less than 1% of modern children under the age of 15 years (Maldonado-Cocco et al., 1980), it has been reported (on the basis of anticipated dam-

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TABLE 1. Frequency (%) of joint involvement in juvenile rheumatoid arthritis

	Juvenile rheumatoid arthritis			Juvenile ankylosing spondylitis		
Joint	Onset[1]	Course[2]	Course[1]	Onset[3]	Course[3]	Course[2]
Shoulder		15	25			10
Elbow		25	40	2	12	5
Wrist	10	50	70	13	21	5
Hand	10		55			
MCP[4]		45				
IP[5] hand		50		11	21	
PIP[6]						
DIP[7]						
Hip	5	10	35	7	21	15
Knee	36	75	90	27	39	75
Ankle	26	30	70	16	29	55
Feet	10		35			
MTT[8]			30	15	23	85
MTP[9]		60				70
IP[5] foot						
Sacroiliac			5	5	13	
Sternoclavicular			3			

[1] Martel et al., 1962.
[2] Burgos-Vargas and Petty, 1992.
[3] Jacobs et al., 1982.
[4] MCP = metacarpal phalangeal.
[5] IP = interphalangeal.
[6] PIP = proximal interphalangeal joints of the hands.
[7] DIP = distal interphalangeal joints of the hands.
[8] MTT = metatarsal tarsal.
[9] MTP = metatarsal phalangeal.

age) in juveniles of archaeological populations, and its possible contribution to our knowledge of past population health has been clearly demonstrated (Buikstra et al., 1990).

Juvenile rheumatoid arthritis (JRA), also referred to as chronic polyarthritis in children, is characterized by synovial hyperplasia, erosive cartilage destruction, and erosion and rarefaction of bone (Gershwin and Robbins, 1983; Pachman and Poznanski, 1993). Juvenile rheumatoid arthritis is traditionally divided into three or four categories: polyarticular, plauciarticular (subdivided into first decade and adolescent onset), and Still's disease (Calabro et al., 1976; Rothschild, 1982).

Still's disease is a febrile disorder often associated with systemic manifestations, including lymphadenopathy, hepatosplenomegaly, and a salmon-colored rash. While not all individuals with Still's disease have arthritis, all individuals with such a systemic presentation are classified as having Still's disease, independent of the nature of any arthritis present. Individuals without such systemic manifestations are divided into polyarticular and pauciarticular varieties, based on number of affected joints. The pauciarticular variety is defined as involvement of less than five joints, while in the polyarticular variety more than four joints are affected. It should be noted that historically (Ansel and Kent, 1977) all forms of JRA were referred to as Still's disease in the early part of this century.

The most commonly affected joints in JRA are knees, wrists, and ankles and the diarthrodial components of cervical vertebrae (Tables 1, 2), associated with decrease in the range of joint motion (Gershwin and Robbins, 1983; Pachman and Poznanski, 1993; Resnick and Niwayama, 1988). Enlargement of or premature closure of epiphyses is found in one-sixth of JRA individuals. Involvement of the mandibular epiphyses often results in a short body and ramus and poorly differentiated condyles. Premature epiphyseal closure or destruction of the epiphysis may lead to ulnar shortening and wrist deviation. One-third have transient phalangeal, metacarpal, and metatarsal periosteal changes, which, in association with endosteal resorption, may produce a rectangular appearance of the bone.

Although JRA was originally divided (Calabro et al., 1976) into three varieties, it was another decade before the confounding

TABLE 2. Characteristics distinguishing juvenile rheumatoid arthritis and juvenile ankylosing spondylitis (figures given are percentages)[1]

	Juvenile, spondyloarthropathy duration					Juvenile, rheumatoid arthritis				
	—	—	6 months	12 months	10 years	—	—	6 months	12 months	10 years
Enthesopathy	77	65	83	89	89	7	20	0	4	4
Polyarticular		30					55			
Pauciarticular		55	54	20	11		45	31	23	16
Sacroiliac/vertebra	65					4				
Lower extremity			83	97	100			76	96	79
Upper extremity			20	37	49			68	80	81
MTP[2]/IP[3] foot			54	63	86			49	64	68
Ankle			49	66	80			51	55	59
Tarsal			71	86	89			1	11	13
Knee			77	100	100			64	83	87
Hip			11	29	83			7	28	37
MCP[4]			0	6	11			39	59	60
IP[3] hands			0	11	23			39	52	52
Wrist			3	6	14			53	61	65
Elbow			3	9	14			23	39	40
Shoulder				20	57				28	37

	Median	Range	Median	Range
At onset	2	1–10	2	1–10
Course	4	1–14	2	1–19

[1] Derived from Hussein et al. (1989), Burgos-Vargas and Petty (1992), and Burgos-Vargas and Vazques-Mellado (1995). Sex ratio (M/F) is 2:1 for JSpA and 1:2 for JRA.
[2] MTP = metatarsal phalangeal joints.
[3] IP = interphalangeal joints.
[4] MCP = inetacarpal phalangeal joints.

concept of juvenile ankylosing spondylitis developed. The latter reflected recognition that some individuals with childhood-onset arthritis had a disorder indistinguishable from that found in adults with ankylosing spondylitis and other forms of spondyloarthropathy (spondylarthritis) (Gershwin and Robbins, 1983; Pachman and Poznanski, 1993; Resnick and Niwayama, 1988; Rothschild and Woods, 1991). Ankylosing spondylitis is a va riety of spondyloarthropathy in which axial disease spreads (in the form of syndesmophytes and zygaphyseal joint fusion) in a bilaterally symmetrical manner from sacroiliac joint through lumbar to thoracic to cervical spine. The diagnosis is one of exclusion among the varieties of spondyloarthropathy (Resnick and Niwayama, 1988). The presence of psoriatic skin lesions in a patient who appeared to have ankylosing spondylitis would generally result in reclassification of the patient's arthritis as psoriatic arthritis.

Differential diagnosis of JRA from juvenile spondyloarthropathy (JSpA) is clinically difficult because most patients with juvenile spondyloarthropathy fulfill (Burgos-Vargas and Petty, 1992; Burgos-Vargas and Vazques-Mellado, 1995; Hussein et al., 1989) the American College of Rheumatology diagnostic criteria (Brewer et al., 1977; Cassidy et al., 1986) for classification as JRA. Criteria for recognition of spondyloarthropathy include axial joint erosion and fusion of axial and/or peripheral joints as well as enthesopathy (Resnick and Niwayama, 1988; Rothschild and Woods, 1991). Limited involvement of metacarpal-phalangeal joints and the frequent presence of enthesopathy and involvement of tarsal joints facilitate recognition of juvenile spondyloarthropathy, at least from an epidemiologic perspective.

The aims of the present paper are twofold: (1) to give a detailed description of the bony lesions associated with a known historic case of Still's disease (the general term in use at that time to identify any form of JRA) and (2) to develop criteria to facilitate distinguishing it from other diseases and from postdepositional erosive processes.

MATERIALS AND METHODS

The skeleton (HTH-2036) of a 7-year-old girl who had been clinically diagnosed in the 1930s as having Still's disease was exam-

TABLE 3. Percentiles and observed range for roentgenographic bones length (cm) of normal girls 7 years old[1] *vs. present case of juvenile rheumatoid arthritis (measurements between epiphyseal plates)*

	Normal						
Bone	10%	25%	50%	75%	90%	Range	JRA[2]
Humerus	18.99	19.60	20.25	20.99	21.71	18.2–23.3	15.4
Radius	13.91	14.41	14.92	15.56	16.06	13.6–17.2	11.4
Ulna	15.41	15.94	16.51	17.09	17.60	15.0–19.1	12.9
Femur	27.01	28.06	29.16	30.15	31.10	26.0–32.2	21.8
Tibia	21.54	22.67	23.56	24.51	25.51	20.7–26.7	18.8
Fibula	21.39	22.39	23.32	24.27	25.09	20.6–26.3	20.3

[1] Maresh, 1955.
[2] Length of bones of the child from the Hamann-Todd collection corresponds to age about 4 years.

ined at the Cleveland Museum of Natural History, where the skeleton is now housed in the Hamann-Todd collection. The autopsy record reported

> marked enlargement of the ankle, knee, and hip joints. All the joint(s) of the fingers, including the metatarsophalangeal [sic] joints, also show marked enlargement. The left thigh is flexed and adducted and cannot be straightened out. The left leg is flexed on the left thigh and cannot be extended. The musculature, especially over the long bones, has undergone extreme atrophy.

The skeleton is complete, with all epiphyses. Macroscopic examination of the skeleton was performed, especially noting articular and periarticular discontinuity, cortical changes, and reactive new bone formation. Marginal erosions were defined if the area of bone excavation (associated with "fronts of resorption" [Leisen et al., 1987]) was limited to the region between the cartilage-covered joint surface and the site of joint capsule insertion (Rothschild and Martin, 1993). Subchondral erosions were identified when the erosive process was predominantely limited to that portion of articular bone originally covered by cartilage. Autopsy x-rays were compared to those taken of isolated bones.

RESULTS

Appendicular skeleton

Upper limb. The shoulder, elbow, and wrist joints are most severely affected. The long bones are light in weight and much reduced in length (Table 3) and breadth, with small epiphyses and premature fusion of the distal humeral and proximal ulnar epiphyses. Muscle markings were minute.

Shoulder joint. Premature fusion of the coracoid process with the scapula is noted, with slight bone reaction on the ventral aspect of the scapula near the glenoid surface. The glenoid itself shows no degenerative changes. The humeral head was completely distorted, secondary to severe marginal and subchondral erosions and bone remodeling (Fig. 1). The proximal metaphysis of the humerus is extremely flat anteromedially and posterolaterally and crescent-shaped in cross-section, probably resulting from limited mobilization and muscular atrophy. Areas of muscle attachment, including the deltoid tuberosity, are nearly invisible. Radiographic findings include generalized osteopenia, intracortical resorption, very thin humeral diaphyseal cortices, marked coarsening of the trabeculae at the distal ends, and marginal and subchondral humeral epiphyseal erosions.

Elbow joint. The distal epiphyses of the humerus are also deformed (Fig. 2). Both the trochlea and the capitulum manifest crenations, subchondral erosion, and bone remodeling. Some of the subchondral erosion is in the shape of a large, deep groove (Fig. 2). The proximal ulna exhibits premature epiphyseal closure, accounting for the short ulna (Table 3). The trochlear notch is almost totally eroded (subchondral) (Fig. 3). Its lateral aspect manifested a deep, elongated, lunate-shaped cavity (Fig. 3). The radial notch is poorly developed. The proximal head of the radius appears normal.

Joints of the wrist. Both ulnar and radial distal metaphyses show slight bone remodeling (Fig. 4). Except for slight porosity, the distal epiphyses of the radius are normal. All carpal bones are deformed and eroded (Fig. 5).

Fig. 1. Superior view of right humeral proximal epiphysis. Severe marginal and subchondral erosions.

Fig. 2. Anterior view of partially fused right distal humeral epiphysis. Subchondral erosion represented by prominant groove on the articular facet. Scale is mm.

Fig. 3. Anterior (**a**) and lateral (**b**) views of right proximal ulna. Subchondral erosions with reactive new bone has destroyed the articular surface.

Joints of the hand. The proximal epiphyses of the metacarpal bones manifest crenation, bone remodeling, subchondral erosive processes, and premature epiphyseal closure. Subchondral and marginal erosions are also present in the distal epiphyses. The diaphyses are thinned and flat, with intracortical bone resorption. Subchondral and marginal erosive processes are clearly seen in the distal end of most proximal and distal

Fig. 4. Anterior-posterior roentgenogram of wrist and hand. Generalized reduction of bone density, with periarticular accentuation. Distal radial and distal ulnar subchondral erosions with increased width of radiocarpal joints space, carpal erosions, subchondral metacarpal phalangeal erosions, broadening of proximal phalanges, and possible proximal interphalangeal erosions.

phalanges. Distal phalanges are reduced in size.

Lower limb. All long bones are much reduced in length, thickness, and density (Table 3), and epiphyses are small (even considering reduction in diaphyseal dimensions). Early fusion of the distal femoral epiphyseal plate is noted. Muscle markings are minute.

Hip joint. The most severe changes were observed in the hip joint, with no pathological changes observed in femoral metaphyses. Both femoral heads are extremely eroded (subchondral), exposing disorganized, sieved trabeculae (Fig. 6), associated with loss of sphericity. The acetabula manifested marked crenation, subchondral erosion, and remodeling (Fig. 7). Radiographic findings include considerable bony demineralization of the alae, femoral head, and femoral shaft, marked coarsening of the trabeculae at the distal and proximal extremities, and subchondral erosions.

Knee joint. Cortical bone at the posterior, medial, and lateral aspects of the distal femoral and proximal tibial metaphyses manifests remodeled perforations, exposing underlying trabecular bone (Fig. 8). Epiphyseal contours show advanced remodeling, marked crenation, and mild marginal erosion. The patellae manifest advanced subchondral erosions and remodeling (Fig. 9). No osteophytes were observed. Roentgenograms reveal thinning of cortical bone, thinning of trabeculae, and marginal erosions.

Joints of the ankle. Erosive processes are manifested in most joints. Slight subchondral erosion is seen on the trochlear surface of the talus. The articular surface for the lateral malleolus on the right talus is slightly eburnated. All articular surfaces of the calcaneus have subchondral lesions. New bone deposition is noted in the region of interosseous talocalcaneal ligament attachment. Subchondral tarsal lesions predominantly appeared in the form of dense, focal pitting on their articular surfaces. Radiologic examination reveal osteopenia and marginal calcaneal-cuneiform erosions.

Joint of the foot. Examination of the proximal articular regions of isolated metatarsal metaphyses reveals clear crenulations with marginal erosion. Marginal erosions are present at the proximal and distal epiphyses of proximal phalanges and proximal epiphyses of middle phalanges, with subchondral erosion of the distal epiphyses of middle phalanges and proximal epiphyses of distal phalanges. Distal phalanges are reduced in size. Radiographs reveal generalized osteopenia and marginal metacarpal erosions. Proximal and distal interphalangeal joint erosions are not well visualized.

Axial skeleton

Skull. The vault of the skull appears normal in size and shape, although the orbits

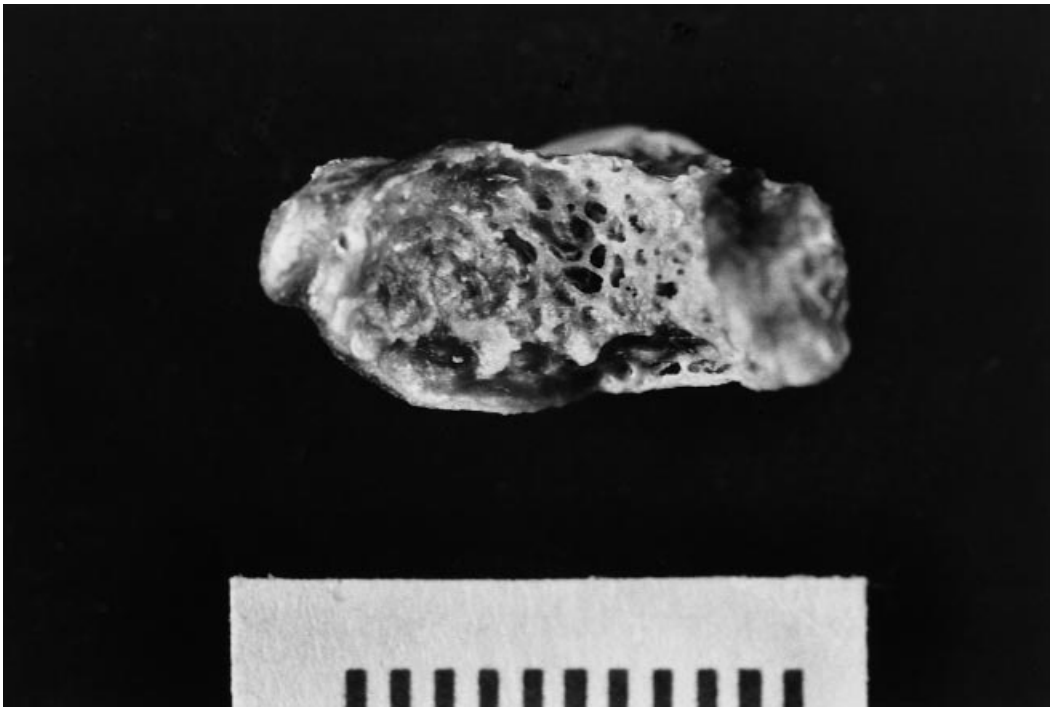

Fig. 5. Surface view of scaphoid. Large erosion. Scale is mm.

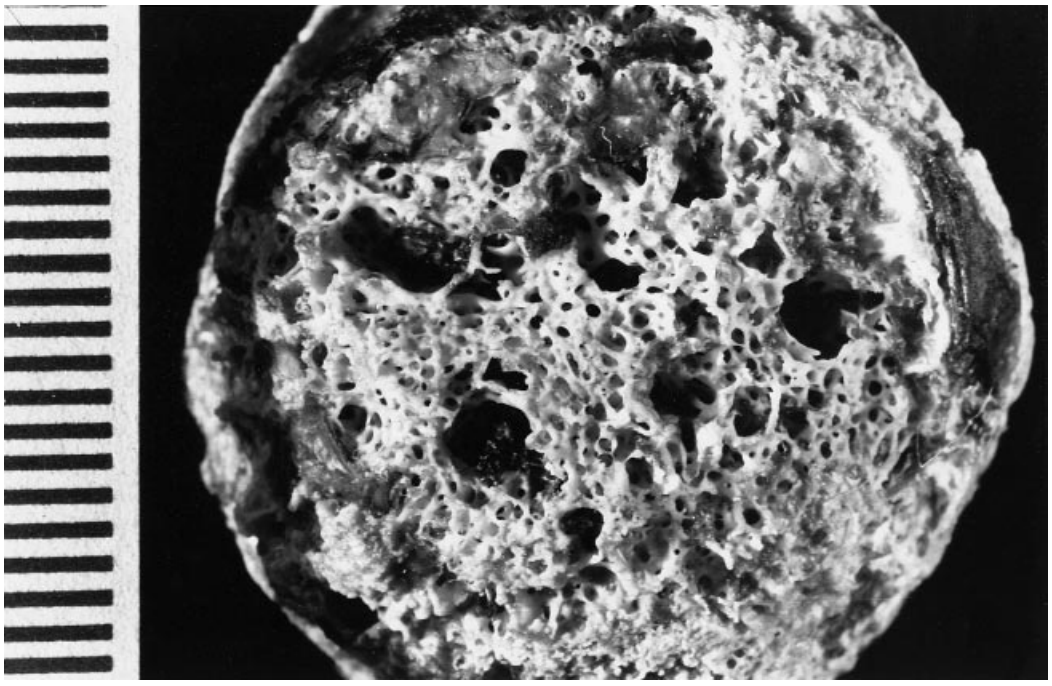

Fig. 6. Superior view of proximal femoral epiphysis. Severe subchondral erosion with remodeling. Scale is mm.

Fig. 7. Acetabular view of right ischium (**a**) and left ilium (**b**). Marked crenation, erosion, and remodeling.

and face are relatively small. Dental status confirmed the age of 7 years. Except for slight porosity of the hard palate and posterolateral parietal and occipital bones, the only pathological changes are in the mandibular fossa. The shape of the mandibular fossa is largely distorted by advanced subchondral erosions (Fig. 10a). The walls of the mandibular fossa show remodeling and perforation. The contiguous root of the right zygomatic

Fig. 8. Posterior view of distal metaphysis of femur and proximal metaphysis of tibia. Thin cortical bone with sieve-like appearance.

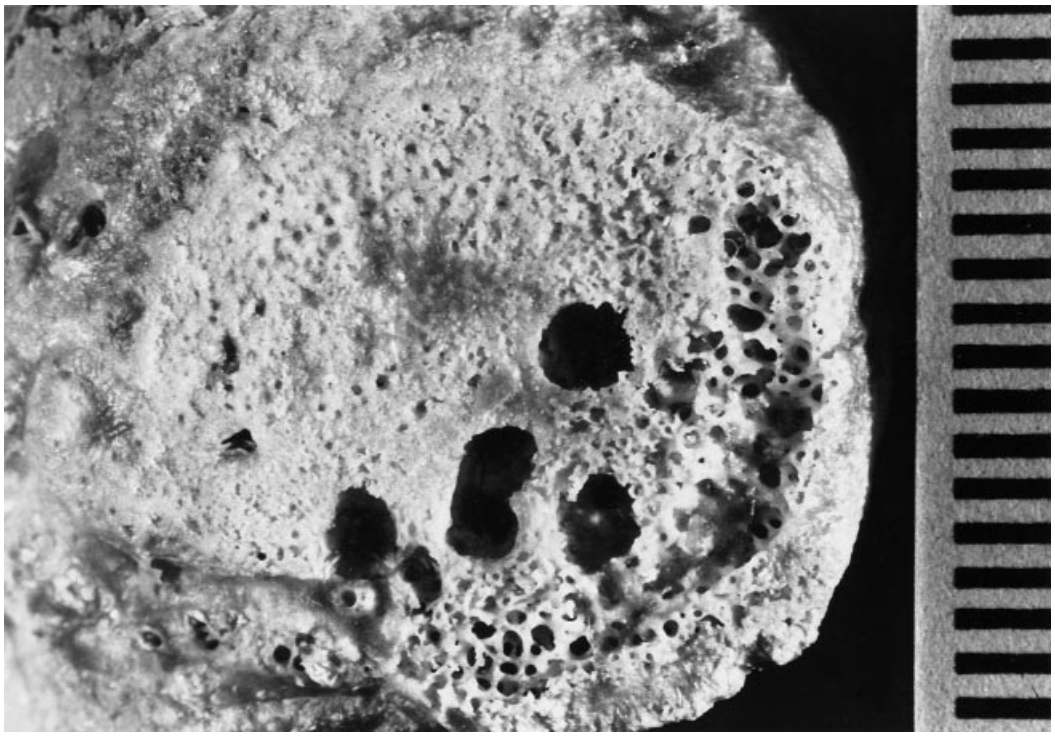

Fig. 9. Posterior view of patella. Subchondral erosion contrast with unremodeled holes (possibly related to a wearing-away process). Scale is mm.

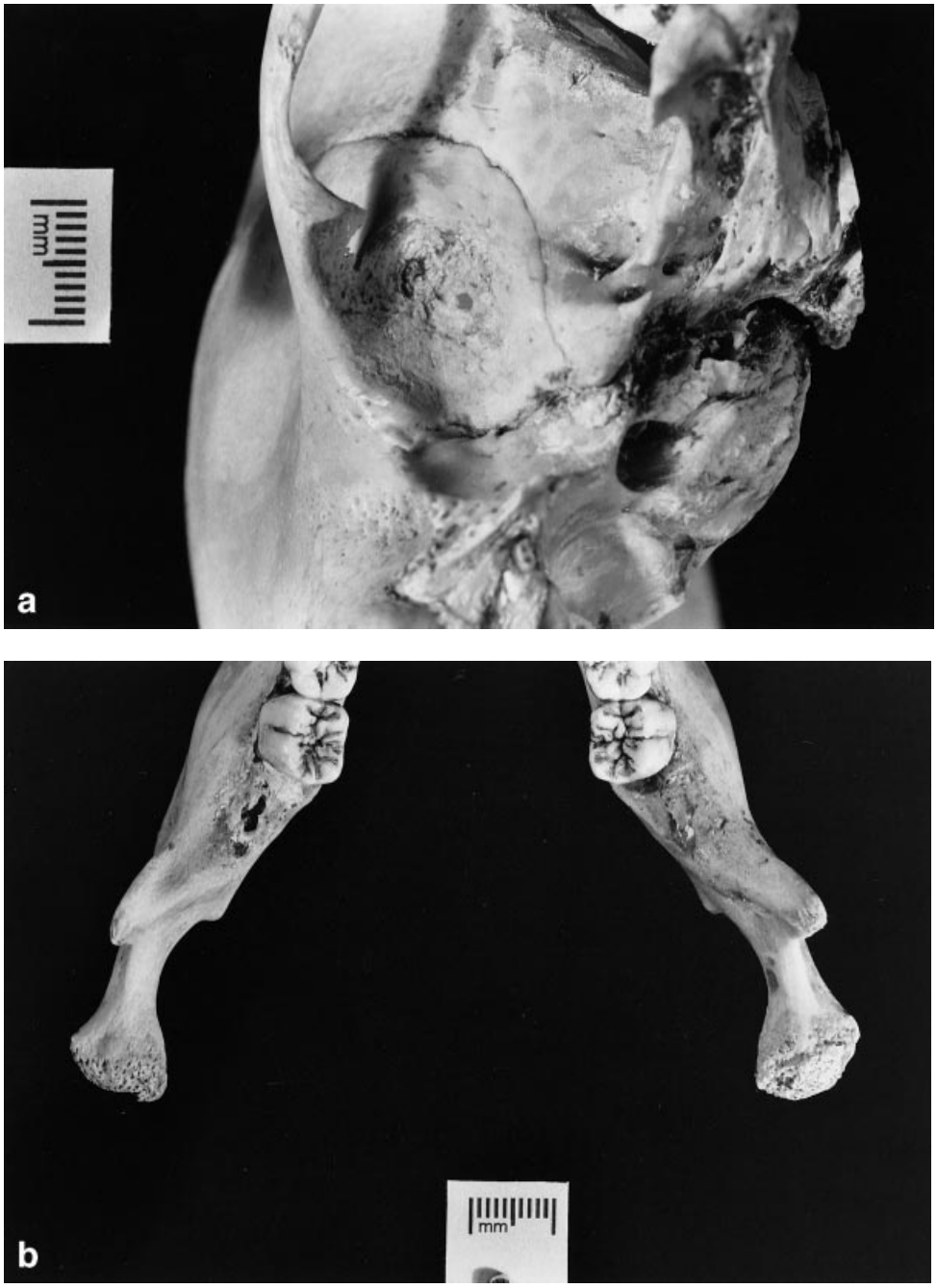

Fig. 10. Advanced lesion in the mandibular fossa (**a**) and corresponding part of the mandible (**b**). Subchondral erosions distort and perforate the mandibular fossa and produced a "pressure" erosion of the contiguous root of the right zygomatic arch, producing a pseudoarthrosis (a). Underdeveloped mandibular condyles (b), associated with disproportionally short, narrow mandibular rami.

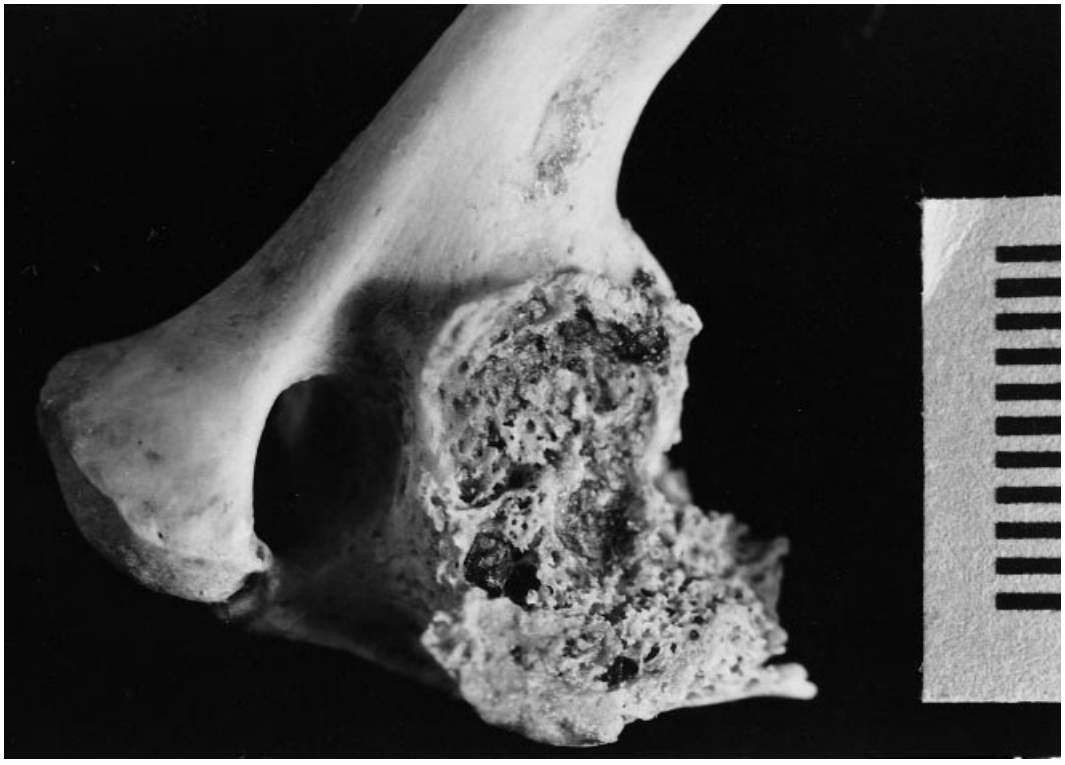

Fig. 11. Superior view of left superior atlanto-occipital joint. Severe subchondral erosion with reactive new bone. Scale is mm.

arch is remodeled, forming an eroded pseudarthrosis. Both mandibular condyles are underdeveloped (Fig. 10b), associated with weakly developed, disproportionally short, narrow mandibular rami.

Vertebrae. Although changes in the articular facets are present throughout the vertebral column, the cervical vertebrae articular surfaces (especially the atlanto-occipital joint) manifest the most severe changes (Fig. 11). The zygapophyseal (articular process) joints of C2 and C3 and C7 and T1 are fused (Fig. 12a,b).

Thoracic cage. The ribs are normal with exception of isolated erosions of the subchondral surface oval facet (for articulation with the vertebral transverse process). The first rib is small. Erosion and bone remodeling are present on the subchondral surface of the sternal end of the clavicle. Slight periosteal reaction is present on the medial clavicular metaphysis.

Pelvis. Both ilia are small, extremely gracile, and osteopenic. The ilia have a reduced typical mushroom appearance, as their anterior border arose almost vertically from the acetabulum to the anterior superior iliac spine. The auricular surfaces are small and porotic and, on the left side, eroded. A large patch of periosteal reaction is present between the iliac tuberosity and the iliac fossa. The iliac tuberosities, ischia, and pubes are small and weakly developed. The ischiopubic rami are atrophied at the junction between ischium and pubis.

DISCUSSION AND CONCLUSIONS

This study describes skeletal evidence of arthritis in a child. The defleshed skeleton of an individual expresses an unequivocal picture of bone affliction characteristic of at least one form of JRA.

The term *juvenile rheumatoid arthritis* originated as an American term to describe

Fig. 12. Lateral view of vertebrae C2 and C3 (**top**) and C7 and T1 (**bottom**). Zygapophyseal joint fusion.

inflammatory arthritis in children (Brewer et al., 1977). Although it was recognized that the disease was quite different from adult rheumatoid arthritis, the term *rheumatoid-arthritis* was then often globally applied to all forms of inflammatory arthritis in adults.

Characteristics helpful in recognition of JRA

Direct observation of the actual impact of documented (in life) JRA on bone suggests characteristics which may be helpful for

recognition of the disease in the archaeologic record:

1. Peripheral articular marginal and subchondral erosions.
2. Axial (e.g., zygapophyseal or sacroiliac) joint erosions.
3. Fusion of axial (cervical zygapophyseal) and/or peripheral joints.
4. Premature epiphyseal closure and/or ballooned epiphyses.
5. Growth retardation with underdeveloped (short and overtubulated) long bones.
6. Short mandibular rami with underdeveloped condyles, producing micrognathia.
7. Demineralization (osteopenia).

Other observations of unclear diagnostic significance include the very small orbits and the eroded deformed ulna. While the severity of the above observed changes will likely vary with age and duration of the disease, the general pattern of the disease may allow diagnosis. These characteristics were present in the presumptive case of Buikstra et al. (1990), further substantiating her diagnostic perspective.

Differential diagnosis

Not all arthritis in children is classifiable as JRA. Some individuals with childhood-onset arthritis have a disorder indistinguishable from that found in spondyloarthropathy (Gershwin and Robbins, 1983; Pachman and Poznanski, 1993; Resnick and Niwayama, 1988; Rothschild and Woods, 1991). Among the characteristics helpful in recognizing juvenile rheumatoid arthritis, only premature epiphyseal closure and/or ballooned epiphyses, growth retardation with underdeveloped (short and overtubulated) long bones, and micrognathia would not be anticipated in juvenile spondyloarthropathy. Differential diagnosis of JRA from juvenile spondyloarthropathy (JSpA) is clinically difficult (Brewer et al., 1977; Burgos-Vargas and Petty, 1992; Burgos-Vargas and Vazques-Mellado, 1995; Cassidy et al., 1986; Hussein et al., 1989) and would not be anticipated to be less so in archaeological material. Because joint distribution helps to distinguish between spondyloarthropathy and rheumatoid arthritis in adults (Resnick and Niwayama, 1988; Rothschild and Woods, 1991, 1992a,b; Rothschild et al., 1990), it was hoped that a similar approach would contribute to distinguishing between JRA and juvenile spondyloarthropathy (Table 2). Limited involvement of metacarpal phalangeal joints and the frequent presence of enthesopathy and involvement of tarsal joints facilitate recognition of juvenile spondyloarthropathy, at least from an epidemiologic perspective. The relative upper extremity sparing seems to be more characteristic of the juvenile ankylosing spondylitis category than of juvenile spondyloarthropathy in general (Burgos-Vargas and Vazques-Mellado, 1995). The latter criteria, however, can be confidently applied only in population studies.

In contrast to the apparent identity of juvenile and adult spondyloarthropathy, juvenile rheumatoid arthritis is not rheumatoid arthritis in a subadult (Resnick and Niwayama, 1988). Although rheumatoid arthritis is by definition a disorder of individuals who have at least achieved the age of 16 years (McCarty and Koopman, 1993; Resnick and Niwayama, 1988), its manifestations are different from those of JRA and JSpA. JRA and JSpA are distinguished from adult rheumatoid arthritis on the basis of postcervical axial joint erosions, axial and/or peripheral joint fusion, the presence of subchondral erosions, and the tendency to distal interphalangeal joint involvement (while sparing metacarpo-phalangeal joints) (Cabane et al., 1990; Elkon et al., 1982; Gershwin and Robbins, 1983; Pachman and Poznanski, 1993; Resnick and Niwayama, 1988; Rothschild et al., 1990; Wouters et al., 1985).

While differential diagnosis could be limited to the above in this individual with the polyarticular variety of JRA, a desire for completeness requires notation of other considerations. The differential diagnosis for the pauciarticular (especially monoarticular) variety of JRA is more complicated. The latter would also include infectious arthritis (suppurative arthritis, osteomyelitis, foreign body, scleroderma, systemic lupus erythematosus, sickle cell anemia, thalassemia, and hemophilia). Suppurative arthritis and osteomyelitis are much more limited in skeletal distribution and are easily recognized because of the associated distortion of bony architecture (Resnick and Niwayama, 1988;

Rothschild and Martin, 1993). The avascular necrosis of lupus is easily distinguished because of the associated collapse of subchondral bone. Hemophilia can produce epiphyseal overgrowth and subchondral erosions, which can mimic those of JRA. It is the associated intraosseous cysts (pseudotumors) which allow hemophilia to be recognized. Sickle cell anemia and thalassemia are associated with bone marrow hyperplasia producing diploic widening, "hair-on-end" calvaria changes, cortical thinning, metaphyseal widening, premature epiphyseal fusion, vertebral end-plate collapse (producing on cross-section an H appearance), obliteration of paranasal sinuses, and the radiologic appearance of a rib within a rib (analogous to osteopetrosis).

Review of the individual in this report reveals characteristics compatible with diagnosis of either JRA or JSpA, precluding absolute confidence in distinguishing between the two phenomena. This is unfortunate, as it compromises recognition of the historical origin of JRA. Although adult spondyloarthropathy is recognizable in the archaeological record as a worldwide phenomenon, significant geographic variation in frequency is noted (Rothschild and Woods, 1992b). Factors determining relative susceptibility (between juveniles and adults) to spondyloarthropathy are unclear in clinical practice (McCarty and Koopman, 1993), and little is known about its pathogenesis (Glass and Litvin, 1980; Miller et al., 1979; Moran et al., 1979; Rossen et al., 1977; Statsny and Fink, 1979). However, study of the archaeological record may prove insightful, as has been the case for rheumatoid arthritis and tuberculosis (Buikstra, 1981; Rothschild et al., 1992). Although the present case study cannot be used to clearly distinguish between JRA and JSpA, the description provided can help to distinguish these diseases from others causing joint pathologies. This is important for those interested in the study of past populations and their health as well as for those interested in the history of disease.

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